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INSTITUTO DE CIÊNCIAS BIOMÉDICAS ABEL SALAZAR
UNIVERSIDADE DO PORTO

Relatório Final de Estágio

Mestrado Integrado em Medicina Veterinária

AN OVERVIEW OF EQUINE ENTERITIS AND COLITIS

Alexandra Xavier de Sousa

Orientador:

Prof.ª Doutora Ana Colette Maurício, DVM, PhD, Agregação

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Summary

The present report is the final assignment of my Curricular Externship, part of the Integrated Master's program in Veterinary Medicine at the University of Porto, under the general theme of Equine Medicine and Surgery. As such, a short review of the cases I had the chance to follow during my stay, from January to May 2017, at the Anglesey Lodge Equine Hospital, in the Republic of Ireland, will be listed. From then on this composition focuses on equine enteritis and colitis, beginning with a bibliographic review, followed by three clinical cases of equine enteritis and/or colitis which I had the chance to follow during my externship.

The bibliographic review will shortly cover possible aetiologies, introduce their pathophysiology, recovery prognostics and diagnostics procedures as well as possible treatments to adopt. In the last section of the paper, after each case is presented, a short discussion will take place.

The goal of this report is to present to my professors, supervisors, and colleagues the work I have done during my externship, by presenting the casuistic I was confronted with and the procedures I had a chance to observe or perform, as well as explore a topic of my interest, both from the academic point of view (with the bibliographic review), as well as the practical aspects of accompanying cases related to this topic.

Acknowledgments

To my mentor, Dr. Ana Colette Maurício for the help and advice I received from the beginning of this project, and to Professor Tiago Pereira for always being available to help, even without being his responsibility.

To all the members of the "turma mais fixolas" of my veterinary class, for true companionship, in good times and outside exam doors or during long days of studying, for the genuine care for each other and in each others' success.

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To Inês, for being a friend like I've never known before.

To Luis, for loving all the right things, and making them even better.

To my sisters who are my best friends and my most fierce defendors, for believing in me more then I do. It's all your fault that I think I'm special.

To my dad who provided for everything and made sure I could focus on what I wanted. To my mom, who gave up everything for us and would give up more if she had it, I hope I'll be more like you everyday.

Index

Casuistic

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**includes foals

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*Differentiated by the therapeutic option taken

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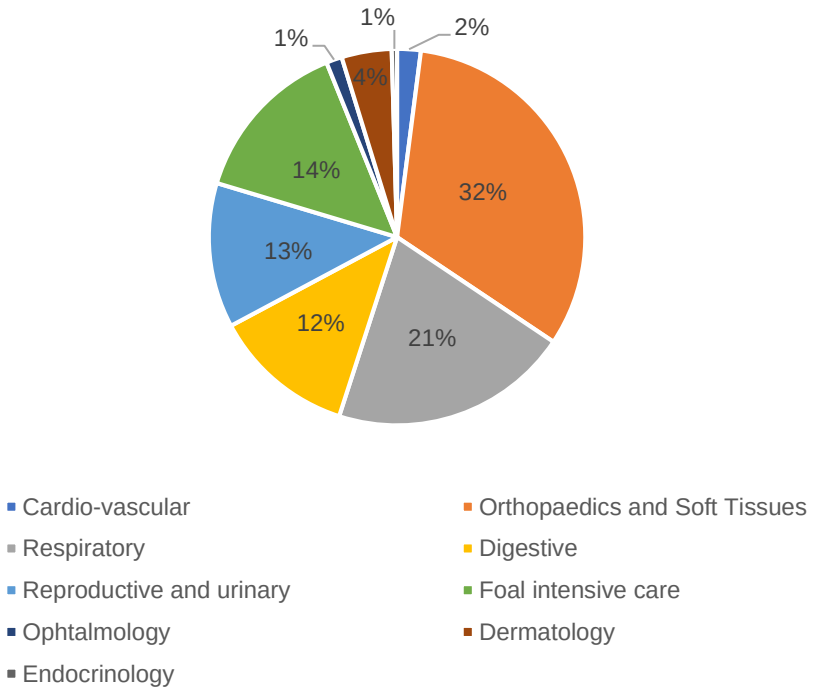
Enterocolitis by *Rotavirus*	3

Endocrinology

Cushing's disease	2

The total number of cases seen was 437.

Graphic 1: Casuistic during the externship by system affected



Procedures*

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Echocardiography	2
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Gastroscopy**	3
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Enema**	3
Joint flushing (under sedation)	2
Euthanasia (chemical)	4

*Procedures seen or performed during the externship. Does not include basic routine procedures such as administering medication (IV, IM, SC, PO or topic), placement of catheters, lameness examinations, monitoring progession of lesions (to tendons and ligaments for instance), taking x-rays or blood samples and changing bandages.
**referes to the number of cases not number of times the procedure was performed as it may have been performed multiple times in one patient.
#complete examination of the horse, lameness exam, lunging and endoscopy, microchip and markings. Radiography and tendon scanning included if any abnormalities found or in certain sales.
¨Includes this procedure done in foals

The total number of procedures I had a chance to see/perform was 549.

For my curricular externship I chose to spend between January and May of 2017 in the Republic of Ireland, at the Anglesey Lodge Equine Hospital (ALEH). The hospital is located in one of the most iconic areas of the equine racing industry, the Curragh, in County Kildare. This natural expanse of grasslands is historically, as far back as the Celtic and Viking invasions, an area

of gathering and horse racing. Today 26% of all racing horses in all Ireland are trained in the Curragh [1], with many racing yards located in its fringes, several gallops, jumps and flat race tracks all within view of the hospital.

Most of the horses coming to ALEH are thoroughbred race horses in training. This makes it so that most cases seen there concern performance problems, such as orthopaedics and soft tissue disorders as well as many upper respiratory conditions. The surgery team at the hospital performs mostly procedures towards resolving those conditions and I had a chance to help in many of them. The surgeons are also involved in the daily running of the hospital overseeing rounds, the daily treatments and any incoming cases.

The ambulatory service provided during my stay was remarkably busy due mostly to the breeding season. The thoroughbred brood mares are monitored closely and treated case by case, both by the veterinarian and the stud farm workers, to ensure the best results are accomplished with only natural covering of the mares. Pregnancy confirmation and monitoring, reproductive disorders as well as cases of lameness, vetting for sales or for the races, and any other disorders or procedures that will not require hospitalization are part of the daily routine of the ambulatory veterinarians.

Some other equines are also seen by the clinicians of ALEH, with jumping and eventing also being popular sports in Ireland and most of its rural population owning ponies or leisure horses without an athletic purpose. Many referral cases are also sent to this hospital, with some horses traveling over three hours to be seen there.

During my stay colitis and/or enteritis was found in three yearlings infected with *Lawsonia intracellularis,* four foals with rotavirus infection, two adult horses with severe diahorrea of bacterial aetiology, one enterocolitis following therapy with NSAID and antibiotics, one anterior enteritis (ileus and severe inflammation) following colic surgery for volvulus and one anterior enteritis in conjunction with hepatic damage. In the last two cases described, as well as one of the *Lawsonia intracellularis* infected individuals, the animals were euthanised due to evidence of extensive damage with poor prognosis, the high cost of treatment and for the sake of the animals' welfare.

Abreviations and Units

CRT – capillary refill time

IV – intravenous administration

IM – intramuscular administration

PO – oral administration

SC – subcutaneous administration

SID – once a day

BID – twice a day

TID – three times a day

PCR – polymerase chain reaction

PCV – packed cell volume

WCC – white cell count

NCC – nucleated cell count

NSAID – nonsteroid anti-inflamatory drugs

SAA – Serum amiloide A

ELISA - enzyme-linked immunosorbent assay

K – Potassium

Na – Sodium

Cl – Chlorum

ºC – degrees Celsius

s – seconds

h - hours

bpm[1] – beats per minute

bpm – breaths per minute

ml – mililiter

cm - centimeter

mm – milimeter

mg - miligram

L – liter

mmol – millimole

UI – International units

Introduction

Enteritis and colitis are the denominations given to the inflammatory disorders of the small and large intestine respectively, caused by a wide spectrum of bacterial, viral, protozoal agents and toxins [2]. The aetiology of these dysfunctions often cannot be differentiated by the clinical findings alone but their clinical management can be quite similar regardless of the diagnostics, the prognosis for recovery, however, can vary greatly [3].

Intestinal disease in horses and foals is mostly suggested by diarrhoea, weight loss, hypoproteinaemia, and abdominal pain [4]. Of all the clinical disorders of the gastrointestinal tract, the ones giving rise to signs of colic are the most common, [3] however, this is only a broad term for a variety of conditions that cause a horse to exhibit clinical signs of abdominal pain, with widely varying aetiologies and severity [4] and therefore too extensive to explore as well in this paper. In enteritis and colitis, the initial stages of disease typically involve localized mucosal inflammation, but this frequently progresses to widespread systemic activation of the inflammatory cascade. Many of the sequelae of these conditions, such as laminitis and multiple organ failure, are related to this systemic inflammatory response [5].

Enteritis and colitis can be acute or chronic regarding their onset of symptoms. For example, in the case of protein-losing enteropathies there will usually be a rapid weight loss and commonly peripheral oedema as a result of hypoproteinaemia [5]. Acute inflammatory intestinal diseases are commonly expressed clinically as diarrhoea, abdominal pain, pyrexia, circulatory failure, and even sudden death [6], cardiovascular compromise and coagulopathy associated with endotoxaemia are also common [7]. A different possible clinical presentation is insidious weight loss, more common in chronic disorders such as neoplastic conditions, sand enterocolopathy and infiltrative lesions (like inflammatory bowel disease or intestinal lymphosarcoma) [4]. Additional clinical signs which may be present in gastrointestinal disease include ileus, anorexia, ptyalism, bruxism, dysphagia and, rarely, skin lesions [3].

Because of the large volume of the gastrointestinal chambers in horses, massive fluid losses can occur in a short time [4]. Thus, diarrhoea in adult horses can be an explosive event with morbidity and mortality exceeding that associated with diarrheal diseases in other animals [4]. The presence of diarrhoea in an adult horse almost invariably indicates large intestinal disease (because the large colon is the primary site of water resorption [7]) whereas weight loss may be associated with disorders affecting either the small intestine or the large intestine or both [3].

There are, of course, other causes for the simptoms we associate with enteritis and colitis which are unrelated to inflammatory disorders of the intestine, this paper may refer to them in passing, in regard with distinction from enteritis and colitis, but does not focus on them.

Bibliografic review

Pathophysiology of enteritis and colitis

Fundamentally, the pathophysiology of enteritis and colitis is inflammatory in nature, and the clinical abnormalities that we associate with these conditions, such as enterogastric reflux and diarrhoea, arise from that inflammatory process [4] [5] [7]. The localized mucosal inflammation, frequently progresses to widespread systemic activation of the inflammatory cascade causing many of the sequelae of these conditions, such as laminitis and multiple organ failure [5]. The other pathophysiological mechanisms of these diseases are all linked to that inflammation.

Inflammation

Inflammation serves a vital role in the host's resistance to infection, as it enhances the directed movement of defensive cells and molecules from the bloodstream to the site [7]. Local inflammation arises as a result of several different insults: direct cellular injury requires that the pathogens gain access to the host's intestinal tissue (as *Salmonella spp.* organisms do); or they may liberate enzymes, toxins, or other substances that damage the host cells; or finally, the inflammatory response alone can be self-harming [5]. The injured cells release pre-formed mediators, responsible for the initiation of a nonspecific inflammatory response. In moderation, the inflammatory response is protective, the excessive and malignant form of the inflammatory response is characterized by the systemic activity of numerous proinflammatory mediators [5].

It is also important to note that while bacterial infection may be responsible for the initiation of an inflammatory response, the inflammatory process itself results solely from the production of endogenous mediators [5].

Pain

Abdominal discomfort is frequently observed in horses with enteritis or colitis [5]. Pain can result from stimulation by local inflammatory mediators in response to mucosal injury. Nociceptors within the gastrointestinal mucosa detect mechanical, chemical, and thermal stimuli

and relay this information to the central nervous system. The accumulation of gas secondary to microbial fermentation of ingested food, which can be combined with impaired passage secondary to ileus, can give rise to significant distension and pain [5]. On the other hand, hypermotility can also be a cause of pain and discomfort due to abdominal cramping [7].

Altered motility

The primary stimuli for hypermotility are inflammatory, and consist of chemical, mechanical, and functional signals related to the injury or dysfunction of the gastrointestinal mucosa secondary to infection or irritation [5]. Hypermotility is commonly present in horses with colitis, leading to shortened intestinal transit times, when combined with increased passive and active fluid secretion into the bowel lumen, the result is increased fecal fluid content and output (increased volume and frequency of defecation) [5]. With diarrheal diseases, the elimination of gut contents is part of the normal host defence mechanism, and thus decreasing motility is not indicated in most cases [7].

On the other hand, gastrointestinal ileus is commonly encountered in horses with enteritis [5]. The presence of ileus in combination with increased secretion of fluid into the small intestinal lumen results in the reflux of large amounts of fluid from the small intestine into the stomach (leading to pain and possible gastric rupture) [8]. The presence of inflammation increases sympathetic tone in the enteric nervous system which suppresses coordinated rhythmic activity of the intestine and results in functional obstruction of the intestine [5]. In some cases of acute colitis, a period of ileus may occur without diarrhoea.

Decreased Absorption

This occurs mostly due to injury of the intestinal cells and diminished intestinal transit times, but several other mechanisms can impair absorption. Dysfunction of the intracellular tight junctions impairs the ability of the mucosal epithelium to retain water and electrolytes after their absorption, as well as disruption of the normal transport mechanisms that move sodium and chloride from the lumen to the interstitium and vasculature [5]. A good example of this are the rotavirus infections, where the structures meant to increase absorption are atrophied, while the crypt cells, which are secretory in function, proliferate [7].

Endothelial Dysfunction

Injury to the intestinal mucosa will increase blood flow and result in a relative increase in hydrostatic pressure within the vasculature, potentiating the flow of fluid into the interstitium and into the intestinal lumen [5]. Another way for this dysfunction to occur is through the loss of albumin from the vasculature to the interstitium in damaged areas, which decreases the intravascular oncotic pressure and allows fluid to leave the vasculature and enter the interstitium as well. The flux of fluid, electrolytes, and protein into the interstitium results in increased interstitial oncotic pressure which leads to the development of interstitial oedema. The fluid and protein within the interstitium may then enter the intestinal lumen by way of the damaged and dysfunctional epithelium, resulting in intestinal fluid losses and protein-losing enteropathy [5].

Increased secretion

The precise mechanisms of secretion in the equine colon are not understood fully but we can divide them into active secretion and passive fluid loss. Bacterial enterotoxins and several inflammatory mediators stimulate active secretion [5]. Passive secretion is the result in changes of hydrostatic pressure in the colonic capillaries, mucosal damage, and loss of tight junctions. This may come to be due to osmotic overload with increase in osmotically active particles within the intestinal lumen (such as magnesium sulphate, carbohydrates or occasionally lipids beyond the organism's ability to absorb or excrete physiologically) [5].

Dietary changes that result in significant shifts in gut flora and changes in fermentation or gastrointestinal diseases that result in malabsorption or maldigestion also may result in an osmotic diarrhoea [7]. Chronic conditions such as congestive heart failure, inflammatory bowel disease, hypoproteinaemia or decreased lymphatic drainage associated with inflammation may increase hydraulic pressure from the blood to the lumen [7].

Endotoxaemia

Endotoxaemia is the term used clinically to describe the clinical signs the equine patient demonstrates when there are circulating endotoxins [7]. Endotoxaemia is of great importance in the hospitalised horse or neonate as it is a major cause of mortality and morbidity [9]. If small amounts of endotoxin enter the portal circulation, they are rapidly neutralised, symptoms arise when the clearing capacity of the liver is overwhelmed with very large amounts - in surgical gastrointestinal conditions, severe colitis or hypovolaemia for instance – or when the endotoxins bypass the portal circulation entering the systemic circulation directly from the peritoneal cavity, pleural cavity or uterus [9].

The clinical features of endotoxaemia include decreased circulating vascular volume, increased capillary permeability, ileus and coagulation disorders, also gastrointestinal stasis and reflux, and maldistribution of fluids with venous pooling, all contributing to massive fluid losses (usually accompanied by significant protein losses too) and hypovolaemia [9].

Specific Diseases

Salmonellosis

Salmonellosis is the disease associated with infection by gram-negative *Salmonella spp* bacteria. The development of the disease depends of the degree of bacterial exposure, the virulence of the *Salmonella spp* organisms (their ability to attach to the mucosa and produce cytotoxins, endotoxins, and enterotoxins), and the susceptibility of the host. *Salmonella* organisms are facultative intracellular anaerobes [3], which allows them to evade some mechanisms of defence. Equine *Salmonella spp* infections are of zoonotic importance [5] [4].

In an infection a local inflammatory response develops first, with an increase of mucosal permeability, secretion of water and electrolytes to the lumen, and intestinal hypermotility, with possible invasion of the mucosa in the ileum, caecum and ventral colon [8]. This loss of fluids, electrolytes, and proteins may be severe requiring aggressive supportive care, it may lead to permanent dysfunction and an overwhelming systemic inflammation [5]. Clinical manifestations therefore range from no clinical signs (subclinical carrier which can shed the pathogen) to acute, severe diarrhoea and even death [4].

Salmonella is found in a low percentage (<1%) of normal horses [8], and even though horses of any age may be affected, foals are significantly more susceptible [3], with stress being a recognized risk factor [8] (as well as recent general anaesthesia/surgery, gastrointestinal disease, antimicrobial therapy, physical exhaustion, dietary change and transportation [8]).

Diagnosis is based on clinical signs, severe neutropenia, and isolation of salmonellae from faeces - faecal samples (more sucessful) or fecal swabs, taken in multiple days - blood, or tissues. [4]. Prevention is difficult, because the organism is present in the environment as well as in the faeces of some healthy animals. Good biosecurity practices, in unconfirmed cases as well, are important, particularly in hospitals. *Salmonella* spp. and *Clostridium difficile* are the most common causes of nosocomial diarrhoea [9].

Clostridiosis

This is the disease most commonly associated with antibiotic-induced, and nosocomial colitis in horses [5]. Clostridial enterocolitis affects both foals and adults and the two primary pathogens concerned are *Clostridium difficile* and *Clostridium perfringens* [7]. These organisms are strict anaerobes and their pathogenicity, since they do not invade the host, depends on production of bacterial toxins.

Cl. difficile produces primarily toxins A and B, the net effect of the activity of these toxins is to induce a secretory diarrhoea with substantial intestinal inflammation [5]. *Cl. perfringens* produces as many as 17 different toxins. The cytotoxins alfa (primary lethal toxin [5]), beta (induces mucosal necrosis, is potentially lethal [5]), epsilon (inactive prototoxin, becomes active and increases intestinal permeability [5]), iota (increases vascular permeability, is potentially lethal) and enterotoxin (highly correlated with pathogenicity, impairs fluid and electrolyte absorption, increases villous blunting and loss of epithelium [5]) are the most important ones.

The clinical signs consist of diarrhoea, fever, depression, mild colic, and dehydration, and in some cases haemorrhagic diarrhoea [7]. The disease can be severe with mortality rates exceeding 50%, with death before treatment is started or even before the onset of diahorrea [8].

Detection of specific clostridial toxins in fresh fecal samples, reflux, intestinal contents, or tissue sample [4] is the best method to make a definitive diagnostic [10]. ELISA test kits can be used at veterinary hospitals now, making rapid diagnosis possible, however one must keep in mind that both pathogens can be present without causing the disease [3].

The most important strategy for prevention of clostridiosis is good farm hygiene, the spores are extremely resilient in the environment and resistant to many disinfectants [7]. Keeping the foaling area and mares as clean as possible during the perinatal period and ensuring rapid ingestion (by stomach tube if necessary) of colostrum within one hour of birth have reduced incidence of disease on contaminated farms [4]. Affected animals should be isolated to limit cross-infection and contamination of pastures and stalls. No proven effective biologic products are available to immunize horses or foals against clostridial enterocolitis [4].

Potomac fever

Potomac horse fever is an acute enterocolitis syndrome producing mild colic, fever, and diarrhea in horses of all ages, as well as abortion in pregnant mares, ventral oedema and laminitis [2]. Clinical signs can be indistinguishable from those of Salmonellosis and other infectious causes of enterocolitis [11]. The causative agent is *Neorickettsia risticii* [11] [8]. The disease is seen in spring, summer, and early fall and is associated with pastures bordering creeks or rivers in the southeast

of the United States of America [8]. The epidemiology has been shown to involve a trematode vector [11].

Clinically ill horses are not contagious and no zoonotic risk is known [11]. A definitive diagnosis should be based on isolation or identification of *N. risticii* from the blood or feces of infected horses by cell culture or PCR. Although vaccination has been reported to protect 78% of experimentally infected ponies, it has been marginally protective in the field [8].

Colitis X

Colitis-X is not a disease but a term used to describe undiagnosed causes of peracute, fatal enterocolitis in horses, characterized by sudden onset of profuse, watery diarrhea and development of hypovolemic shock [4]. Typically it involves single isolated cases rather than outbreaks [3]. *Clostridium perfringens* and *Clostridium cadaveris* are the most suspected causative agentes, *Salmonella spp* and *Escherichia coli* have also been suggested, and it has recently become apparent that many *C. difficile*–affected adult horses present with classic clinical signs and lesions of colitis X [12].

The typical clinical pathology signs include edema and hemorrhage in the wall of the large colon and cecum on necropsy, PCV over 65%, neutropenia, with metabolic acidosis and electrolyte disorders [2]. Death may occur within 3 hours of onset of clinical signs or in less acute cases, within 24–48 hours [4].

Duodenitis proximal jejunitis or anterior enteritis

This condition affects the upper small intestine, causing ileus [8] and resulting in distention, abdominal pain, gastric reflux, and increased peritoneal fluid protein concentration (without a significantly elevated nucleated cell count) [7]. These horses often have evidence of multiple organ involvement and can have hepatic changes thought to result from ascending infection [7]. The exact cause of this malady is yet to be elucidated, however, organisms such as *Salmonella* spp, *Clostridium perfringens, Clostridium difficile* as well as *Fusarium* spp have been implicated, and so have alterations in diet, a high percentage of grain in the feed and several other factors [7] [3].

A tentative diagnosis may be reached on the basis of clinical progression and response to gastric decompression, along with the clinicopathologic changes, but a definitive diagnosis can only be made by gross examination of the duodenum and proximal jejunum at surgery or at necropsy (the ileum and large colon usually are determined to be grossly normal) [7] [8].

Due to the difficulty in diagnosing this disorder, distinction between a small intestinal obstruction and this pathology plays a major role, since the first may warrant surgical intervention [7] [8].

The main signs found in animals with this pathology that are differenciating from small intestine obstructions are:

- the abdominal pain typically subsides after gastric decompression [7];
- larger volume of gastric reflux (4 -20L with each decompressive effort) [7];
- on rectal palpation they have less distension [7];
- often present with fever [7] [8];
- typically very dehydrated [7];
- protein concentration in the peritoneal fluid is often higher then 3.5 g/dl [7];
- electrolyte alterations, prerenal azotemia, and elevated hepatic enzymes are common [7] [8].

Medical treatment of horses with these pathology is advocated over surgical management, which has lower survival rates and does not decrease nasogastric reflux which is a primary therapy goal [8]. Survival rates have been reported to be anywhere from 25% to 95% (regardless of therapy) with recurrence of the disease being rare [8].

Viral Enterocolitis

Rotaviral enterocolitis is the most widespread and significant form of viral enterocolitis in foals worldwide [7] but rarely affects adult horses [4]. Affected foals range from 2 days of age to 6 months of age [5], the younger the more severely affected [7], and the diahorrea can persist for weeks [4]. Rotavirus is highly contagious and outbreaks may develop rapidly [5].

Rotavirus results in villous atrophy and villous blunting in the duodenum and jejunum, this impairs intestinal absorption and leads to increased secretion of fluid and electrolytes [7]. Lactase becomes deficient, so lactose passing into the large intestine induces an osmotic diarrhea [4]. The initial clinical signs are anorexia and depression, with profuse watery diarrhea occurring shortly thereafter [8]. Infection is ultimately self-limiting [5] [7].

Diagnosis is made by identification of the virus in the feces by electron microscopy or commercial immunoassay kits designed for detection of human rotavirus, with clinically normal individuals also shedding the organism. The survival rate is typically very high, with clinical signs ranging from mild to severe [8], and effective vaccination of pregnant mares available [7] [8].

A definitive role for **adenovirus** has not been established in the foal, however, adenovirus is a common co-isolate from foals with rotaviral diarrhea [7].

There have been recent outbreaks in adult horses of **coronavirus** [8] with clinical signs including anorexia, lethargy, fever and sometimes colic and diahorrea. Occasionally, rapid progression leads to death (or euthanasia), but most cases resolve with supportive care [4]. Healthy foals have been found to be infected by equine coronavirus as a single infection, whereas in sick foals it has been found exclusively in association with other coinfecting agents [8].

Intestinal parasites

Both large and small strongyles have been incriminated as a cause of chronic diarrhea in horses and foals [4]**.** As control of the large strongyles has improved, the small strongyles (*Cyathostominae*) have assumed their current role as the primary parasitic pathogen of the horse [5]. In **cyasthostominosis** the disease is associated with the larval stages of this parasite which invade the gastrointestinal mucosa, encyst and when they emerges as a adults an associated interstitial edema, inflammation and eosinofilic infiltration result, disrupting normal motility [5]. It causes weight loss, marked and progressive, diahrroea of sudden onset, possibly severe which usually becomes chronic, colic is usually mild and may not be presente, and ventral oedema is variable [3]. Many horses with cyathostominosis go undetected and untreated since until their sudden eruption there are no clinical manifestations [5].

Large strongyles that are pathogenic in horses include *Strongylus vulgaris* (the most important [7]), *Strongylus edentatus*, and *Strongylus equinus* [7]. Strongylosis causes weight loss, poor body coat condition, colic may be severe and occur in recurrent bouts, diarrhoea (less frequente [7]), anaemia and lethargy [3]. Migration of larvae through the intestinal wall affects myoelectrical activity and motility, affecting retention of ingesta and absorption of fluids [7].

The prognosis is good for general strongylosis and fair for larval cyathostominosis (50% survival rate) [3]. The diagnostic is by identifying large numbers of cyathostomins or eggs, in the case of strongyles, in diarrhoeic faeces or rectal swab (in cyathostominosis, excretion of *Salmonella* spp. should be regarded as secondary) [3]. Preventive measures include reducing exposure of susceptible individuals, and instituting proper deworming schedules [7].

Protozoal Enterocolitis

The primary protozoal enterocolitis in the horse is **Cryptosporidiosis**, caused by *Criptosporidium parvum* [5], which exhibits little or no host specificity, is highly resistant to antimicrobial agents, and has the potential to cause autoinfection [3]. Equine cryptosporidiosis

occurs primarily in young foals, with or without immunodeficiency [4]. This organism is zoonotic, with cases reported among humans handling horses and calves that are shedding cryptosporidial organisms [5].

Giardiasis has been reported in a limited number of cases as a cause of intermittent diarrhea in horses. However, *Giardia* can also be found in the feces of a small number of healthy horses and is rarely recognized as a cause of diarrhea in horses [4].

NSAID-Associated Right Dorsal Colitis

Toxicity of the nonsteroidal anti-inflammatory drugs involves the renal system as well as the gastrointestinal system, and all segments of the gastrointestinal tract can be affected [4]. The toxic potential of these drugs arises inherently from their mechanism of action, an indiscriminate suppression of prostanoid production [5]. The maintenance of normal mucosal function and health requires the presence of prostaglandins E, F, and I whose primary function appears to be the maintenance of normal mucosal blood flow and tight junction functionality [5]. Disruption of these functions by inhibiting the activity of both cyclooxygenase-2 (associated with inflammation), and cyclooxygenase-1 (responsible for the production of these prostaglandins) results in mucosal barrier dysfunction and injury [5]. This leads to the signs of nonsteroidal colitis, namely secretory diarrhea with hypoproteinemia, in association with mucosal ulceration, neutrophilic inflammation and colon wall edema [3]. Right dorsal colitis (ulcerative inflammatory bowel disorder) has been associated with the administration of NSAID particularly in horses treated when dehydrated or toxaemic [13]. Phenylbutazone is the nonsteroidal drug that has been associated with most reported cases of right dorsal colitis. A recente rise in number of cases associated with flunixin meglumine may be a result of an increase in this drug's use in order to avoid the well documented toxic effects of phenylbutazone [5].

Inflamatory Bowel Disease

This is a collection of diseases including granulomatous enteritis, lymphocytic-plasmacytic enterocolitis, multisystemic eosinophilic epitheliotropic disease, and idiopathic focal eosinophilic enterocolitis [4]. Disease is characterized by infiltration of the small and large intestine with inflammatory cells (lymphocytes, plasma cells, macrophages, and eosinophils). Malabsorption and a protein-losing enterocolopathy are the result, with or without diarrhea. Inflammatory bowel disease should be considered in the differential diagnosis of horses with weight loss, recurrent colic, or hypoproteinemia [4]. The prognosis is grave, various medical treatments have been tried

with limited success, surgical removal may be successful if only a limited and accessible section of the bowel is affected.

Neoplasia

Squamous cell carcinoma of the stomach and the alimentary form of lymphosarcoma are the most common forms of neoplasia involving the gastrointestinal tract in horses [4]. Chronic weight loss may be the primary clinical sign. Chronic diarrhea and hypoalbuminemia may develop when lymphosarcoma has infiltrated the wall of the intestine. Because the incidence of gastrointestinal neoplasia is low, other causes of weight loss should be investigated first [4].

Non inflamatory/non intestinal aetiologies

Nonintestinal causes of chronic diarrhea include congestive heart failure and chronic liver disease. The diagnostic approach to these cases is aimed at differentiation of infiltrative diseases of the intestine from physiologic causes of diarrhea [4]. Other exemples include grain overload, thromboembolic disease of the colon, peritonitis, renal failure, numerous toxicoses like cantharidin, salt poisoning, amitraz, propylene glycol, arsenic, mercury, organophosphates, avocado, thorn apple, potatoes, mycotoxicoses, hyperlipidosis, among many others [4].

Antibiotic-associated enterocolitis

Certain antibiotics, such as trimethoprim-sulfonamide combinations, erythromycin, penicillins, tetracyclines, clindamycin, and lincomycin, are associated with enterocolitis syndromes in horses. In some cases, such as those seen with trimethoprim-sulfonamide combinations, the geographical incidence of antibiotic-associated diarrhea appears to differ markedly [3].

Grass sickness

Grass sickness is a neurological disease associated primarily with degeneration of neurons of the autonomic nervous system, in particular the enteric nervous system (dysautonomia), which is typically manifest by gastrointestinal signs. Horses are afebrile and show tachycardia, ileus, and colic, distended loops of small intestine and an impacted large colon in the more acute cases [4]. It is hypothesized but not yet proven that the aetiology is a toxicoinfection with *Clostridium botulinum.* Incidence is highest in spring and in horses between 2 to 7 years old. Acute and chronic forms are recognized depending on whether death occurs within 24 hours or 7 days [4]. The disease

is common in regions of the UK and parts of northern continental Europe, and a virtually identical condition occurs in South America [3] .

Sand enteropathy

Sand ingested from the environment accumulates within the large colon and can result in mucosal irritation [7]. Large amounts of sand accumulation can result in nonstrangulating obstruction. Horses consuming sand may have signs of diarrhea or poor body condition. Mucosal abrasion can result in endotoxemia [14].

Proliferative Enteropathy

The causative agent of equine proliferative enteropathy is *Lawsonia intracellularis*, an obligate intracellular organism [5]. Reports of equine proliferative enteropathy have increased in recente years with weanling foals between 4 to 6 months of age being most commonly affected [7]. Infection leads to dramatic proliferation of immature crypt enterocytes, resulting in loss of the normal villous epithelium and gross thickening in the small intestine [5] [15]. Lesions can also include focal erosions or ulcers, most commonly found in the ileum and terminal jejunum [7]. This alters absorption of nutrients and fluid secretion by disrupting the architecture of the villi and by altering the maturation of epitelial cells into absorptive cells. The clinical signs include weight loss, peripheral edema, diarrhea, and colic. Hypoproteinemia leading to peripheral edema are characteristic findings, as well as substantial fluid and electrolyte deficiencies [5]. Secondary complications such as gastric ulceration, bronchopneumonia, and parasitism may occur concurrently [15]. *Ante mortem* diagnosis through serologic analysis of *L. intracellularis* antibodies and PCR analysis of feces can be performed [7].

Reported survival rates for affected foals/weanlings with appropriate treatment range from 81 to 93%. These results reinforce the fact that, with early detection and appropriate therapy, most foals should survive. The gastrointestinal abnormality that is characteristic of this disease is not permanent in nature [3].

Inflammation is not responsible for the clinical signs since characteristically there is little or no inflammation present [5], therefore this not an enteritis or colitis but is instead an infectious proliferative disease that mimics enteritis.

Relatioship with the Liver

Colic and diarrhoea, although suggestive of intestinal disease, are also common clinical signs of hepatopathy. Conversely, hyperammonaemic encephalopathy, although suggestive of hepatic disease, is also reported secondary to intestinal disease. Furthermore, cases of primary intestinal disease are occasionally seen that develop clinical or clinicopathological evidence of hepatopathy during hospitalisation [9].

Diagnostic Approach

Not every horse with diarrhoea has enteritis or colitis, horses that have had an exploratory laparotomy in the last 24 hours may have diarrhoea as a direct result of the procedure, for instance [16]. When looking for the aetiology of gastrointestinal disease important information may be collected particularly from the anamnesis, physical examination and additional exams such as rectal palpation, abdominal ultrasound and blood analysis (haematology and biochemistry) [7]. Examination of patients with disease of the gastrointestinal tract must include evaluation of the metabolic and cardiovascular status of the patient, because acute conditions can lead to endotoxemia and sepsis [7].

Anemnesis and patient description

Age: foals and yearlings are more susceptible to gastrointestinal infections such as rotavirus, *Lawsonia intracellularis* and salmonellosis [3]. Larval cyathostominosis is more common in horses less than four years old [3]. Chronic inflammatory bowel disease (CIBD) and intestinal neoplasia are more common in horses greater than ten years old. [3]

Number of cases: multiple cases are suggestive of an infectious cause, such as salmonellosis in acute enterocolitis cases, or larval cyathostominosis and proliferative enteropathy in cases with weight loss and diarrhoea. [3]

Duration/progression: acute systemic illness due to endotoxaemia occurs in various enterocolitides and also peritonitis. On the other hand, cyathostominosis cases tend to have sudden onset, but rarely have marked systemic illness, and tend to have a timecourse of a few weeks. CIBD and intestinal neoplasia are generally insidious, with a duration of weeks to months [3].

Recent treatment with nonsteroidal antiinflammatory drugs (NSAID), antimicrobials or anthelmintics are relevant as each may cause gastrointestinal disorders [7], as well as changes in diet and inadequate deworming [7].

Traveling/geographic localtion: several of these diseases have specific geographical areas of manifestation or higher prevalence.

Phisical Examination

There are virtually no characteristic physical findings in these pathologies, and the clinical approach is to rule out other disorders and undertake clinicopathological investigation relevant to intestinal dysfunction [3]. Colic, reduced borborygmi and cardiovascular compromise consequente to endotoxaemia, occur in acute enterocolitis and peritonitis which must be differentiated from a strangulating intestinal obstruction or a non-strangulating infarction, among others. Musculoskeletal signs which might be useful for differential diagnosis may be detectable in certain disorders primarily manifest as gastrointestinal in nature such as the characteristic stance of a horse with grass sickness (the four feet placed close together under the trunk) versus a laminitic stance [3].

Abdominal auscultation is particularly useful for assessing the motility of the large intestine [7]. Typical progressive borborygmi can be heard every 3 to 4 minutes on both sides of the abdomen [7]. Less frequent progressive sounds may indicate a pathologic condition of the large intestine or may result from anorexia, nervousness, or pharmacologic inhibition of motility. Absolute absence of any auscultable borborygmi suggests abnormal motility and indicates ileus resulting from a serious pathologic condition [7]. One can detect sand or gravel in the large intestinal ingesta by auscultation listenigng for sand or gravel particles grinding together [7].

Abdominal Percussion can reveal gas in the large intestine by producing a *ping* when simultaneous digital percussion and auscultation are performed over a gas filled viscus. This technique is particularly useful in foals, ponies, and miniature horses because of the limitations of rectal palpation [7].

***Per rectum* palpation**

Palpation per rectum should be performed to confirm that horses do not have a different primary disease (e.g., small colon impaction) or a secondary lesion (e.g., colonic displacement or impaction). It can be a definitive diagnostic tool for some clinical problems such as pelvic flexure and small colon impactions for instance. It can also be used to provide additional information regarding the part of the gastrointestinal tract affected, including distended small intestinal loops or large colon distention [14].

Abdominal ultrasound

A systematic approach to evaluating the abdomen will optimize the chances of identifying abnormalities, especially when the clinical signs and physical examination do not localize the problem to a specific abdominal structure [14]. Intestinal motility, distention, contents, and wall thickness should be evaluated [14]. Peritoneal fluid is often pocketed in the cranioventral abdomen and it is important to evaluate its volume and echogenic characterisitcs. Normal small intestine has minimal visible structure [17]. In foals, special attention should be paid to determine if gas is present within the intestinal wall, suggestive of necrotizing enterocolitis, the large and small colon should be evaluated for the presence of meconium. The umbilicus should be evaluated as should the bladder. The renal pelvis may be dilated without cortical thinning in foals receiving intravenous fluid therapy.

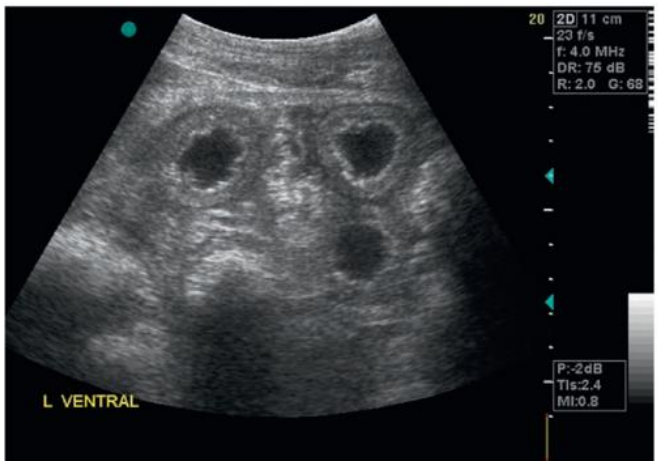

Figure 2 - Trans-abdominal ultrasonography showing tickened loops of small intestine due to inflammation. In ***Atlas of Equine Ultrasonography*** *[17]*

Sand along the mucosal surface appears as hyperechoic pinpoint material and can cast acoustic shadows. Right dorsal colitis can be diagnosed by identifying a thickened right dorsal colon wall in the right 10^{th}–14^{th} intercostal spaces just ventral and medial to the liver [14]. Gas produces a series of horizontal parallel reverberations on ultrasound [17].

Transrectal ultrasonographic examination may be useful in further characterizing abdominal masses, rectal masses, or intestinal surfaces [17]. It is importance to perform serial sonographic evaluation over time to determine if anatomic changes have occurred, such as position of the colon or amount of intestinal distension for instance. [17]

Nasogastric intubation

Nasogastric intubation and reflux is a diagnostic procedure but a therapeutic procedure as well. One should evaluate the following caracteristics in the reflux obtained:

- Volume: if in excess of 2 L, this is suggestive of a gastric outflow problems, most often due to ileus, proximal enteritis, or a small intestinal obstruction [3];

- Color: green/brown is considered normal, yellow is consistent with reflux from the small intestine, and orange/red may be consistente with hemorrhagic enteritis [3];
- pH: normal pH is 4–6, if the fluid is from the duodenum and jejunum the pH will increase to 6–8 [3].
- Samples of reflux can be used for *Salmonella* spp. culture and toxins screening for *Clostridium difficile* [3].

Abdominocentesis

Abdominocentesis is a means of assessing intestinal damage, or in horses with a fever of unknown origin to diagnose peritonitis. It can also be useful in horses with unexplained weight loss, inappetence, and chronic intermittent colic for detection of neoplastic cells [8].It is very useful in the assessment of acute abdominal disorders but of limited value in chronic conditions. Peritonitis can be confirmed by detection of peritoneal fluid white cell count above 5×10^9/L, total protein over 10 g/L and alkaline phosphatase of more than 250 IU/L. Intestinal neoplasia and chronic inflamatory bowel disease rarely exfoliate nucleated cells into peritoneal fluid in detectable numbers. [3] Other findings of biochemestry analysis include: increased lactate, decreases in fluid pH, and abnormal color and/or turbidity which are most strongly correlated with ischemic strangulating lesions [8].

Transudates have low protein concentrations and nucleated cell count (NCC). The most common causes of transudates are the peracute/acute phase of any lesion causing decreased venous/lymphatic drainage in the portal system (e.g., volvulus/torsion, neoplasia, and granuloma), lymphatic obstruction, acute uroabdomen, and protein-losing nephropathies and enteropathies [8].

Blood haematology and biochemestry

There are no specific markers of gastrointestinal disease, the most relevant analyses to measure are total protein, albumin, globulin, alkaline phosphatase, fibrinogen and a complete blood count (CBC) and differential [3]. In acute gastrointestinal disease, hydration status, acid-base balance and electrolytes (K, Na, Cl) disturbances should be assessed, together with renal function (creatinine, urea and phosphate). The level of the abnormalities found relate to the severity of chronic enteropathies.

The most common alterations in haematology are a high PCV, and leucopenia or neutropenia [7]. The packed cell volume is often monitored during gastrointestinal disease to monitor fluid status, but should always be considered along with other parameters such as blood

lactate and urine output [9]. Increases in PCV are attributable to dehydration and/or splenic contraction (splenic contraction is epinephrine-mediated and may occur with excitement or pain [14]).

Infectious and inflammatory disorders of the intestine can cause increases or decreases in the peripheral white cell count [9]. In general, diseases that result in a large release of endotoxins, such as salmonellosis, result in decreased white cell count, due to margination of the neutrophils in capillaries. There may be a brief period of leukocytosis before the leukopaenia in these horses, and a leukocytosis may return during the recovery phase. In clostridial diarrhoea, there is generally a moderate leukocytosis. [9]

The typical regenerative response to anemia in horses is macrocytic and normochromic [18]. Reported cases of larval cyathostominosis have been associated with microcytosis. Nonregenerative anemia due to anemia of chronic disease may also be normocytic and normochromic.

Common biochemical laboratory findings for enteritis and colitis include hypoproteinemia/ hypoalbuminemia, hyperlactatemia, azotemia, hyponatremia, hypochloremia and metabolic acidosis [7]. Electrolyte disturbances are extremely common in gastrointestinal disease, and these should be monitored closely during acute disease and in horses after colic surgery, to allow prompt supplementation [9]. Coagulopathies in hospitalised horses are highly associated with endotoxaemia and therefore, with severe gastrointestinal disorders. Monitoring the coagulation system is advised in at risk patients [9]

Gastrointestinal disease can result in secondary damage to other organs, such as the kidney and liver. Intermittent monitoring of enzyme activities and metabolite concentrations associated with these organs can allow early identification and treatment of secondary organ damage or dysfunction [9].

Therapeutic approach

The treatment of enteritis and colitis in most cases is supportive, as many of these conditions have poorly defined etiologies (e.g., duodenitis proximal enteritis-anterior enteritis) or are caused by agents that are not likely to respond to specific therapy (e.g., rotaviral diarrhea, salmonellosis). The principles of treatment of these diseases are often similar since they produce similar patterns of clinical and clinicopathologic alterations.

Fluid therapy

Substantial losses of fluid from the circulating volume usually necessitate supportive fluid therapy [5] and given the active secretion of fluid and electrolytes into the lumen of the intestine, IV fluid volumes of up to 40–80 L/day may be necessary [4]. A fine balance between fluids lost and fluids administered must be maintained to limit overhydration, which may lead to increased third-space accumulation of fluid within the gastrointestinal tract [8]. Accompanying losses of protein may necessitate colloid therapy as well. Electrolyte derangements are often present, requiring that supplementation be provided either enterally or parenterally [5]. Other deficits should be determined by serum biochemical analysis and supplementation with sodium chloride, potassium chloride, calcium gluconate, magnesium sulfate, and occasionally sodium bicarbonate may be indicated [4].

Equine plasma may be administered to correct hypoproteinemia and to supply coagulation factors and, depending on the source of the plasma, specific antibodies for endotoxin and *Salmonella*. Colloidal plasma substitutes such as hetastarch may be necessary to maintain oncotic pressure in horses with substantial protein loss into the GI tract. These colloidal plasma substitutes may be less expensive and better tolerated than equine plasma in some horses. Often, equine plasma and colloidal plasma substitutes are both used in horses with hypoproteinemia due to colitis. [4]

Anti-inflamatory

Anti-inflammatory therapy is indicated in many of these conditions to address both the local and systemic components of the inflammatory response [5]. NSAIDs such as flunixin meglumine, help counteract the effect of endotoxin, control pain, and possibly help prevent laminitis. Serious adverse effects, such as gastric and colonic ulceration and renal nephrotoxicosis, can result from NSAID treatment, so the minimum effective dosage should be used [4]. Nonsteroidal antiinflammatory drugs such as flunixin meglumine (0.25 to 0.5 mg/kg, IV, every 8 hours) or firocoxib (0.1 mg/kg, IV, every 12 hours) should be administered for their antipyretic and analgesic effects, as well as to attenuate the effects of endotoxemia on the cardiovascular system [8]. Flunixin meglumine is thought to be a more effective analgesic, but given its contribution to delayed mucosal healing and the possibility of nephrotoxicosis in volume-depleted animals, administration of firocoxib should be considered [8].

Antimicrobial

Antimicrobial therapy is not indicated in all cases [5], for instance in adult horses with salmonellosis it is controversial and does not appear to alter the course of the colitis or decrease

shedding of salmonellae [4]. However, antimicrobials are fundamental in treating most aetiologis related to enteritis and colitis, either being targeted at the main cause of disease or targeting secondary infections or the unwanted proliferation of enteric microbes. An effective treatment of Lawsonia is the administration of a macrolide (erythromycin or clarithromycin) with rifampin or a tetracycline (oxytetracycline or doxycycline) for 3 weeks [8]. In virus enterocolitis, very young foals may benefit from intravenous plasma administration and broad spectrum antimicrobial coverage to limit bacterial translocation [7]. For Clostridiosis, a specific treatment with an antimicrobial, metronidazol, which is both an antibiotic and an antiprotozoal, is available and effective [8]. Oxytetracycline is the drug of choice for treatment of Potomac horse fever [7].

Antitoxic

Low-dose polymyxin B (6,000 units/kg, bid) has been advocated to bind circulating endotoxin. In controlled trials, polymyxin B ameliorated some of the known effects of endotoxemia in horses. Antimicrobial doses of polymyxin B are substantially higher than the dose used to bind endotoxin and may be nephrotoxic, however, low-dose polymyxin B therapy is unlikely to be nephrotoxic in adequately hydrated horses receiving IV fluids [8].

Other therapeutic options

Disruption of normal segmental and/or progressive motility may necessitate decompression by **nasogastric intubation** or surgical intervention, and prokinetic therapy may be of benefit [5]. Control of abdominal discomfort is often accomplished by decompression of the stomach by nasogastric intubation, in the case of enteritis, or by the passage of diarrhoea in horses with colitis.

The degree of overt abdominal pain usually subsides with appropriate supportive care in these conditions. The low-grade discomfort commonly seen with enteritis and colitis following the initial acute stages is controlled best by administration of nonsteroidal anti-inflammatory medications or by administration of other types of **analgesics**, such as opioids or alpha-2 adrenergic agents [7].

Decreased voluntary feed intake, or forced withholding of feed, often necessitates nutritional support [5]. If **parenteral feeding** is required, the use of **gastrointestinal protectants** (eg, biosponge, bismuth subsalicylate, activated charcoal) may be beneficial since they bind bacterial toxins [4]. Sucralfate can be administered orally in these cases as a gastrointestinal protectant and to discourage bacterial translocation [7].

If a Rotavirus infection is present, an **enteral rest** of 1 to 3 days' duration may be useful, as the damage to the small intestinal villi results in a relative deficiency of lactase, resulting in

exacerbation of the diarrhea from accumulation of osmotically active lactose molecules within the intestinal lumen [5].

Antihelmintics may be administered even if the presence of parasytes is not yet confirmed as therapeutic measure but also a diagnostic one. In cyathostominosis, administration of an **anthelmintic** to horses with a heavy load of encysted larvae may also cause rapid larval death and acute and often severe inflammation similar to natural emergence [8].

The yeast *Saccharomyces boulardii* produces a protease that specifically degrades *C difficile* toxins A and B. Thus it has been shown to be protective in clostridial diarrhea in other species, and there is some evidence of beneficial effects in the treatment of horses with colitis [4].

Clinical Case I

Patient description: 2 year-old throroughbred filly, in training for racing.

Anemnesis: The filly was first examined at a race yard (on 28/03/2017). She had a history of three episodes of watery diahrroea in the past two and a half months, with no other symptoms, both resolved withing 24 hours without treatment. The filly had been dewormed recently and no other horses were affected. A blood sample was collected and revealed elevated urea (13.4 mmol/L). The body temperature was also elevated (38.9ºC). The decision was made to bring the horse to que hospital to beguin fluid therapy.

Phisical examination upon arrival: the filly was alert and responsive, was seen urinating normally and no diahrroea was observed (had a dirty tail). Heart rate was 36 bpm¹, respiratory rate was 20 bpm, rectal temperature was 38.3 ºC (10ml of Finadyne® were administered before arrival). The mucous membranes were slightly congested and dry, with CRT less then 2s. Gut sounds were present and normal in all quadrants, and no digital pulse was found.

Further examination: on rectal palpation a general distension of the gut was the only palpable abnormality, with no individual strands of small intestine palpable or any large colon abnormalities. An abdominal ultrasound was performed and revealed distension in the small intestine, with fluid filled loops visible and moving well, with a slight increased thickness of the walls in some areas (6 mm). No abnormalities where found in any other structures. Another blood sample for haematology, biochemistry and SAA examination were performed and found only the same value of elevated urea as well as fibrinogen (3.04 g/L). Elevated creatinine kinase was also

found, but it was discarded as clinically relevant since this is a race horse in training, which explains a slightly elevated marker of muscle damage.

The filly was placed in isolation in an intensive care unit. A faecal sample was collected at the beginning of the examination and sent to be examined by the Irish Equine Center (IEC) for microbiology (faecal culture and antibiogram).

Presumptive diagnostic: enteritis of unknown cause

Therapeutic Plan:

Therapy	**Dose and frequency/rate**	**Objective**
Hartmanns® (ringer's lactate solution)	Continuous infusion at the rate of 0.6L/h for 4 days	Fluid therapy for hydration
Colvasone® (dexamethasone sodium phosphate 2 mg/ml)	20ml IV SID	Corticosteroid anti-inflammatory
Finadyne® (flunixin Meglumine 50 mg/ml)	10ml, IV, SID (only on the day of admission before arrival)	NSAID and anti-pyretic
Biosponge® (di-tri-octahedral smectite)	100g diluted in 1L of water PO BID	Intestinal protectant
Transfaunation	5L, PO, BID (added on the third day)	Transplante of healthy intestinal fauna
Gastrogard® (omeprazol 37%)	one full syringe (6.16 g) PO, SID	Treating and preventing gastrointestinal ulcers
Feed to be withheld and allowed dry hay only	*Ad Libitum*	For intestinal rest
Flagyl® (metronidazol 250mg/tablet)	36 tablets PO, BID (added on the third day)	Antibacterial and antiprotozoal

Table 1 showing the therapeutic options taken, their frequency and administration, and their target in the therapeutic plan.

Case development: The filly continued with diahrroea, normal gut sounds, no signs of dehydration or digital pulse and normal temperature for the next three days (no abnormalities on any physical examination) without any more Finadyne® being administered. The therapeutic plan presented was followed and blood samples were taken at least once a day and hematology and biochemistry including urea and creatine, electrolytes, SAA and lactate, were measured each time, and showed elevated fibrinogen (3.04-2.53 g/L) and urea (9.5-13.9 mmol/L), having both become normal in two days (on 30/03/2017). The filly had good appetite and the faeces gradually improved in consistency and on the fifth day of hospitalization were normal.

Results of the faecal sample culture and sensitivity exams: moderate growth of *E.coli*, with heavy growth of coliforms in general as well as alpha haemolytic *streptococcus*, and light growth of beta haemolytic *streptococcus.* Of all the antibiotics tested, these agents were all sensitive to amoxiciclin (with or without clavulanic acid added), cefuroxime, cefquinome, ceftiofur and enrofloxacin. With all other antibiotics tested at least two microbes were resistant. No *salmonella spp* or *campylobacter spp* were found on the specific growth plates.

Definite diagnostic: enteritis of unknown origin/mixed infection of opportunistic microorganisms
Prognostic given: good for a full recovery with adequate antibiotic covering.
Given these results the treatment plan remained unaltered, sweet feed was re-introduced slowly. The filly was discharged after 8 days of hospitalization (on 04/04/2017), with instructions to be kept apart from other horses until her next examination in two weeks. No medication was prescribed for at home treatment. Exercise was to be mild at first and increased over two/three weeks, however an injury in the right hind limb kept the filly in box rest for three weeks.

Discussion of clinical case I

As mentioned before, a definitive cause of diarrhea can be determined in under 50% of cases [4], and this case is one of those. None of the most common pathogens responsible for enterocolittis was present, nothing in the history of the animal pointed to any specific cause, there were no other animals affected and with only non-specific treatment, aiming at the symptoms presented, the disorder was resolved.

The main findings were the presence of diahrroea, elevated blood urea and fibrinogen, and pyrexia. In a case of intestinal disorder, absortion of nutrientes is compromised, this leads to an insuficiente intake of dietary energy, and the need to break down body tissues for energy, this will elevate blood urea [5]. Liver or kidney damage could also lead to this finding, however no other indications of the involvement of these organs was found. The elevated fibrinogen tells us there is a chronic inflamatory process occuring (the SAA, a marker of acute inflammation, remained unaltered), it is a mild increase which are more commonly associated with viral disease and non-septic tissue inflammation (eg. neoplasia) [7], however dihydration alone can cause small increases of fibrinogen such as this one [19]. On the contrary, the presence of pyrexia suggests an infeccious aethiology, but can in fact be found in a number of noninfectious disorders including immune-mediated and neoplastic disorders [9]. Dehydration would also help explain the rise in blood urea concentration and the dry and congested mucous membranes of the filly on arrival. Horses with enteritis or colitis often present with dehydration, which occurs secondary to fluid losses in the form of diarrhea or enterogastric reflux, in combination with decreased voluntary fluid intake [5].

Since the aetiology is unknown, the treatment is aimed at the symptoms presented, and Table 1 associates the diferent therapeutic actions taken and the goal kept in mind when administering them.

The population of bacteria found to be proliferating in the intestines of the filly were opportunist agents, part of the normal comensal fauna, and commonly found in the environment.

Therefore, the cause of disease is though to lay somewhere else, the most likely explanation being that these microbes simply proliferated in an already altered envirnment. The only antibiotic used for treating this filly was metronidazol. Metronidazol is effective in the treatment of enteric obligate anaerobic bacterial infections, and some protozoa, however it is not effective against facultative anaerobes or obligate aerobes [20], which is why it is comonly used in combination with other antibiotics [8]. Metronidazol may confer additional benefits from its antiinflammatory effects and immunosuppressive actions [8].

Since the horse was comfortable with no signs of colic, no nasogastric tube was passed to relieve pression, despite the evidence by ultrasonography of fluid accumulation in the small intestine, and there was no need for analgesic drugs or sedation.

Three months after these events the filly was back in full training, with no more signs of recurrent pathology, with good prospects of winning its debut race.

Clinical Case II

Patient description: 11-month-old filly, kept on the field with other yearlings during the day and brought inside at the end of the day.

Anamnesis: On the 6th of February 2017, the filly was very dull in the field so an ambulatory veterinarian was called to see her. She had a high temperature of 39.7ºC, heart rate was 60 bpm¹, respiratory rate 12 bpm, mucous membranes were pink and CRT was 3s, gut sounds were present but reduced in all quadrants. A blood sample was taken and therapy with Engemycin® (oxytetracycline 100mg/ml, 15 ml IV) and Pro-bute® (phenylbutazone 200mg/ml, 4ml IV), both SID, was started. For the next three days, the filly was monitored at home and the same therapy continued, she seemed to have improved, with no more episodes of pyrexia, eating and drinking well, and no abnormalities in the clinical examinations. As the filly was not excreting faeces for the duration of the three days, a dose of mineral oil (10ml/kg, PO) was given on the third day. The next day the filly was very dull, with rectal temperature of 39.7 ºC, heart rate of 60 bpm¹, respiratory rate 12 bpm, mucous membranes where congested (dark pink) and dry. A blood sample was taken and it revealed a high urea (16.3 mmol/L) and PCV (55.7%), with decreased total protein (29 g/L) and albumin(16 g/L), so the decision was made to admit the filly in the hospital to begin intravenous fluid therapy (on 09/02/2017).

Phisical Examination upon arrival: the animal was very dull, rectal temperature 39.0 ºC, heart rate was 66 bpm[1], respiratory rate was 12 bpm, and gut sounds were normal on the left quadrants but reduced on the right quadrants. A catheter was placed and fluid therapy started straight away.

Further examinations: On rectal examination, a few distended small intestine loops could be felt, but no other alterations. Ultrasound examination of the lungs found no abnormalities. On the abdominal ultrasound several loops of small intestine were visible, with reduced motility and thickened wall (6-8mm), some areas of small intestine more caudally were also observed to look normal and moving. No more abnormalities were found. A faecal sample was collected and sent for faecal culture, egg count, PCR for *Lawsonia intracellularis*, and specific cultures for *Salmonella spp* and *Campylobacter spp.* Other abnormalities found in blood haematology and biochemistry were elevated SAA (157.6 mg/L) and fibrinogen (3.6 g/L), electrolyte alterations (elevated phosphorus 3.24 mmol/L and decreased calcium and sodium, 2.43mmol/L and 130.7mmol/L respectively), a high percentage of lymphocytes (49%) and low neutrophils (48%).

Presumptive diagnosis: Protein-losing enteropathy

Therapeutic Plan:

Therapy	**Dose and frequency/rate**	**Objective**
Hartmanns® (ringer's lactate solution)	Continuous infusion at the rate of 0.6L/h for 4 days	Fluid therapy for hydration
Colvasone® (dexamethasone sodium phosphate 2 mg/ml)	18 ml, IV, SID	Corticosteroid anti-inflammatory
Marbocyl® (marbofloxacin 10%)	6ml, IV, SID	Antibiotic
Engemycin® (oxytetracycline 100mg/ml)	15ml, IV, BID	Antibiotic
Voluven® (6% hydroxyethyl starch, in 0.9% sodium chloride solution)	2 bags of 500ml, IV, SID (only on the first day)	Colloid, for rapid plasma volume expansion and oncotic support
Gastrogard® (omeprazol 37%)	one full syringe (6.16 g) PO, SID	Treating and preventing gastrointestinal ulcers
Finadyne® (flunixin meglumine 50 mg/ml)	5ml, IV, SID or BID depending on pyrexia	NSAID and anti-pyretic
Flagyl® (metronidazol 250mg/tablet)	26 tablets *Per Rectum*, BID (added on the third day)	Antibacterial and antiprotozoal
Hiperimune plasma	1L bag, IV, every 48 hours for 4 days	Colloid for volume expansion and also to repair protein loss and prevent coagulopaties
Panadur® (fenbendazole 10%)	23ml, PO, SID (2 days	Dewormer
Protium® (40 mg/vial of pantoprazole)	3 vials of 40mg, PO, SID (2 days	To treat and prevent gastrointestinal ulcers
Buscopan® (hyoscine butylbromide 20 mg/ml)	20ml, IV, SID (only during colic fit)	Analgesia for abdominal pain (antispasmodic)
Clexane® (enoxaparin sodium, one vial has 300 mg/3 ml)	1.5 vials, SC, SID (started on the second day after thrombophlebitis)	Profilaxy for coagulopaties, and clot organization and resolution

Table 2 showing the therapeutic options taken, their frequency and administration, and their target in the therapeutic plan.

Case development: on the day of admission the filly showed some signs of colic during the evening (flank watching and pawing), the gut sounds were normal in all quadrants, but no faeces

passed. She had a high temperature during the night (38.9ºC) and received Finadyne® (according to treatment plan). The filly also developed during the night an oedema of the neck and head (PCV 48%, total protein 1.8g/dl) and a lameness (4 out of 5, obviously lame walking) on the right hind limb with a swollen and warm fetlock joint. She remained dull and with congested mucous membranes.

A specialist of internal medicine came to see the filly the next day (10/02/2017) and examine her more thoroughly. After an ultrasonography of the neck, the oedema of the head was though to be caused by a lymphatic obstruction, as a thrombos was completely obstructing the left jugular vein, and an early thrombosis was found around the catheter site on the right jugular vein (the catheter was removed and a new one placed in the left cephalic vein). An endoscopy was also performed, oedema and inflammation throughout the pharynx were noted but not interfering with swallowing (tested with water) or breathing. Therapy with Clexane® was started, as well as local thermotherapy. The abdominal ultrasound was repeated, thickened non-motile loops of duodenum (6.6-9cm in diameter) filled with fluid were still present, with loss of motility seen throughout the small intestine. The differential diagnostics included duodenal stricture, abdominal lymphoma or infection with *Lawsonia intracellular*is and, whichever the diagnostics, the prognostics for survival was estimated to be approximately 40% given the damage to the intestine already done and the clinician's experience. A sample was also collected from the synovial fluid of the right hind fetlock, it had a nucleated cell count of 3000 cells/µL and total protein of 20g/L so it was thought to confirm an immune mediated synovitis. The next day the filly seemed to improve at first with no episodes of pyrexia, a slight improvement in the facial oedema, willing to eat and no other abnormalities in her physical examinations. The joint swelling went down and the lameness improved (1 out of 5) and one pile of soft normal droppings was seen. In the afternoon however, the animal showed signs of colic - pawing, lying down, looking at the flank - with increased heart rate (between 80 and105 bpm[1]) and respiratory rate (40 bpm) with laboured breathing. Finadyne® was administered (6ml, IV) and the animal responded well looking comfortable and eating an hour later with normal heart and respiratory rates. The rest of the therapeutic plan was continued. Over the next two days (11-12/03/2017) the animal was pyrexic (between 38.5ºC and 41ºC) with short lived response to Finadyne®, with intermittent signs of colic (despite normal gut sounds) and no droppings passed. The heart rate was consistently high (50-80bpm[1]), the respiratory rate was normal, and she had decreased apetite. The blood analysis of those days showed for the first time increased white blood cells count ($9.6x10^9$/L), and the abnormalities seen before remained present. The results of faecal sample examination came back, no eggs were seen, no salmonella or

campylobacter found in their specific growth plates and the only findings were a heavy growth of alpha haemolytic *Streptococcus spp* and moderate growth of *E.coli* and yeasts. The PCR analysis of a faecal sample **confirmed an infection with *Lawsonia intracellularis.***

A day after (the 13/02/2017) a new abdominal scan showed the small intestine with no motility, wall thickness of over 1 cm, and a large pocket of peritoneal fluid free in the abdominal cavity, suggestive of peritonitis, a sample was taken and the fluid was very thick opaque and yellow. Analysis of the peritoneal fluid revealed a white cell count of 48×10^9/L which confirmed peritonitis and suggested a compromised gut. The owner was presented with the options of operating immediately for an exploratory laparotomy (with poor prognostic and possible euthanasia on the table) or euthanizing the animal, on welfare grounds. The animal was taken to surgery and the findings were consistent with a severe enterocolitis with necrotic areas of the gut, the decision was made to euthanise the animal. Images and the *post mortem* exam conclusions can be foun in Anex II.

Two more horses from the same breeding farm were affected with *Lawsonia intracellularis* (all the yearlings from the farm were tested), both were much less severely affected than this filly, they came to the hospital for treatment later, a similar treatment plan to the one presented for this filly was followed and they both survived and recovered fully.

Discussion of clinical case II

The main clinical findings in this case were pyrexia, a high PCV, congested mucous membranes with increased CRT, tachycardia, high blood urea with decreased total protein and albumin.

Dehydration caused by the diahrroea explains alone most of these findings. The elevated PCV and increased CRT show that this is a moderate to severe case of dehydration, accounting partly for high blood urea as well as for tachycardia and elevated fibrinogen. In this infiltrative disease, alterations in mucosa are extensive and compromise the absortion of nutrientes, leading to protein degradation by the host and increased blood urea [5]. No biochemical markers of damage in any other orgasn, besides the gastrointestinal tract, were found.

The elevated fibrinogen and SAA show there is an acute inflamatory process occuring [7] (fibrinogen is usually used as a chronic marker, however its concentration can increase up to 10 times within 24 hours of stimuli [19]). The presence of high and recurrent pyrexia suggests an infeccious aethiology or the presence of toxicity in the blood, linked to the proliferation of gastrointestinal pathogens.

The thrombophlebitis is a common complication of intravenous fluid therapy, causing mechanical blockage, and may be a nidus for infection. Edematous occlusion of the nasal passages can result from bilateral jugular vein thrombosis and can be fatal [5], hence the need for an endoscopy. Coagulopaties, such as disseminated intravascular coagulation, have been associated with local and systemic infections, protein-losing enteropathy, and acute gastrointestinal disease [21]. No specific tests for coagulation functions or evaulation of coagulation were performed in this case.

Colloid therapy is an essential component of treating cases of hypoalbuminemia such as this one. Plasma stabilizes the oncotic pressure by directly providing albumin and has the advantage of also providing coagulation factors, antithrombin, and immunoglobulins. Hydroxyethyl starch is a commonly used synthetic colloid, but it may cause coagulopathies [8].

Elimination of the bacteria necessitates an antimicrobial with good intracellular penetration, this include oxytetracycline [8]. Metronidazol PO, aimed at controling the gastrointestinal proliferation of other pathogens can be combined with oxytetracycline and has the additional benefit of providing local gastrointestinal antiinflammatory and immunosuppressive actions [8].

The diagnostic of immune-mediated synovitis may have been precipitated in this case. It is a reportedly rare consequence of infections in equines, when present it is usually in the form of polysynovitis and citology of the cells found in the synovial fluid (non-degenerated neutrophils, plasma cells and lymphocites) would confirm it but was not performed. However, supporting the imune-mediated origin, is the fact that in a septic synovitis one would expect higher protein content and nucleated cell count in the fluid, and no growth of pathogens was seen in the standard plates incobated with the sample (blood agar incubated aerobically or anaerobically at 37°C). The joint was not treated with antibiotics or any anti-inflamatory drugs except the systemic drugs in the treatment plan.

Most horses with *L intracellularis* infection survive if appropriate treatment is initiated early in the course of disease [8]. Despite not being an inflamatory disorder but rather an infecious infiltrative disorder, the clinical manifestations are very similar, and this is a case representative of a protein-losing enteropathy, as well as an emerging disease in Ireland, which I had never seen before.

Clinical Case III

Patient description: One month old foal, kept in the field with the mare during the day and taken inside at the end of the day.

Anemnesis: the foal had intermittent diahrroea at home for the past two weeks, was nursing well and active with no other symptoms, however he became very dull suddenly and was brough to the hospital because he had a fever (39.2ºC) and was very dull with signs of dehydration (dry and pale mucous membranes). Three more cases similar to this occurred within the same week, all in the same farm, in foals between two weeks and two months old. The owner had decided not to vaccinate for rotavirus infection this year.

Phisical examination on admission: heart rate 80 bpm[1], rectal temperature 38.1 ºC, respiratory rate 20 bpm, mucous membranes pale and dry, CRT under 2 s. Gut sounds present and normal in all quadrants, very dull attitude and dirty tail and back legs with watery faeces. No other abnormalities were found.

Further examination: A blood sample was also collected for haematology and blood biochemistry analysis which revealed increased urea (157 mmol/L) and creatinine cinase (320 IU/L) levels, as well as elevated fibrinogen (5.99 g/L) and SAA (115.6 mg/L). Electrolytes were also altered with diminished levels of calcium (2.75 mg/L), sodium (127.7 mmol/L) and potassium (2.32 mmol/L). No changes were found on haematology except an elevated percentage of monocytes (6.5%).

Diagnostic: Rotavirus infection (confirmed by PCR)

Therapeutic plan:

Therapy	Dose and frequency/rate	Objective
Hartmanns® (ringer's lactate solution) supplemented with potassium chloride and glucose (50% solution)	1 L every 6 hours, with 10ml of potassium chloride and 50ml of glucose	Fluid therapy for hydration, correcting electrolyte concentrations and supplementing glucose intake
Marbocyl® (marbofloxacin 10%)	2ml, IV, SID	Antibiotic
Cobactan® (cefquinome 4.5%)	4ml, IM, BID	Antibiotic
Flagyl® (metronidazol 250mg/tablet)	5 tablets, PO, BID	Antibiotic and antiprotozoal
Gastrogard® (omeprazol 37%)	2ml (for 100kg foal) PO, SID	Treating and preventing gastrointestinal ulcers
Antepsin® (sucralfate 1g/5ml)	20ml solution PO, TID	Treating and preventing gastrointestinal ulcers
Separation of mother and foal with a cage inside the box	For the first 4 days of hospitalization, interrupted by a few minutes of feeding every two hours	For intestinal rest and also to avoid the accumulation of lactose in the intestine contributing to diahorrea

Table 3 showing the therapeutic options taken, their frequency and administration and their target in the therapeutic plan.

Case development: The foal was in the hospital for a week. On the second day at the hospital the foal developed a thromboflebitis in his right jugular vein, where the catheter was placed, therefore the catheter was moved to his left cephalic vein. A bandage was applied to his neck and thermotherapy performed. The therapeutic plan was followed with no alterations during his stay. The abnormalities found in the biochemistry and haematology exams presented before gradualy resolved, as did his diahrroea. No other symptoms developed so the foal was discharged 7 days later with instructions to be kept apart from the other foals in the breeding farm for another week, and for at home therapy, Doxicycline generic (10mg/kg PO, SID) capsules were prescribed. The other cases were also positive for rotavirus, the farm was considered endemic and the owner begun vaccinating all the pregnant mares immediately and testing all the remaining foals.

Discussion of clinical case III

In rotavirus infections, intestinal absorption is impaired and there is increased secretion of fluid and electrolytes to the intestinal lumen [7]. This leads to the dehydration present in this case, which is in turn relates to the fever, tachycardia, and dullness, increased blood urea and electrolyte disturbances.

Elevated creatinine cinase levels, having not been accompanied by an increase of AST or other liver specific biochemical markers, suggests acute rabdomyolisis [22]. If muscle necrosis has ceased, a return to normal values occors within 3–7 days [22], as was the case here. Skeletal muscle represents the largest intracellular fluid compartment in the body, rhabdomyolysis thereby significantly alteres the electrolyte composition of extracellular compartments [22]. In haematology, erytrocites are microcytic relative to adult reference intervals until 9 months to 1 year of age [18], therefore this was a clinical finding I did not consider relevant.

Elevated fibrinogen and SAA confirm the presence of inflammation, and consecutive measures of these two markers where used to monitor the progression of recovery and efficacy of treatment.

The separation of the foal from his mother was to prevent him from nursing *ad libitum*. Nursing was allowed every two hours for a minute (the time and frequency were progressively increased as the foal improved). This is to give a chance of rest to the intestinal tract, and prevent the accumulation of lactose, since in this disorder lactase often becomes deficient, and

accumulation of lactose induces an osmotic diarrhea [4]. The foal was provided hay *ad libitum* once he showed signs of improvement and had good apetite.

There is no specific treatment for the virus, and as such the treatment was aimed at treating, and preventing, secondary infections to wich foals are particularly susceptible, with broad spectrum antibiotics and antiprotozoal agents.

Vaccination of pregnant mares is an effective means of prevention [7] [8], however it can be quite costly, needing three administrations of the vaccine during the pregnancy to be effective [8]. The owner of this farm had problems with this infection in the past, but given the high survival rates [8] and the mild clinical signs he took a chance. The farm was severely affected by the disease this time around, with two other foals requiring hospitalization (one of them in two different occasions), and many more foals being treated for less severe manifestations at the farm.

The subject of rotavirus was of particular interest to me because it is representative of my work with foals during my stay at Anglesey Lodge Equine Hospital, requiring around the clock care and close monitorization with complications arising frequently, as well as being a not so common pathology since prevention brought down the incidence rates.

Conclusion

The goal of this report was to present the work I have done during my externship and exploring a topic of my interest as well as a few clinical cases related to it.

My time at the Anglesey Lodge Equine Hospital allowed me to observe a lot of different cases, both at the hospital and in ambulatory services, to participate in several surgeries and learn about the veterinary reality in Ireland.

The bibliographic review shortly covered the most common aetiologies, as well as diagnostic procedures and treatment options. The definitive determination of the etiology underlying enteritis and colitis can be challenging. For some syndromes, no definitive etiologic agent has been identified, requiring that diagnosis be based on clinical signs and non-specific clinicopathologic abnormalities. Ultimately, obtaining a definitive diagnosis is of most importance when dealing with potentially infectious agents, in order to minimize the risk of dissemination of the disease throughout a farm or hospital population [5]. Yet, treatment of these disorders is very similar, allowing supportive therapeutic management despite the lack of a definitive diagnosis [4].

In the last section of this paper, after each case was presented, a short discussion took place, referring pertinent therapeutic options as well the relevance of each case to the accurate representation of the disorder and of my externship.

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Anex I

In order to facilitate a more practical approach to the aetiologies of enteritis and colitis here are some simple tables that help with organizing differential diagnostics and the steps until a diagnostic is reached.

Differential Diagnoses for Chronic Diarrhea in Adult Horses	
Cause of Diarrhea	**Major Diagnostic Test(s)**
Chronic salmonellosis	Fecal culture or polymerase chain reaction, culture
Sand	Fecal sedimentation
Parasitism (strongylosis, cyathostomiasis)	Fecal egg count, empirical deworming
Nonsteroidal anti-inflammatory toxicity (primarily right dorsal colitis)	History and supportive clinicopathologic findings, ultrasonography, exploratory surgery with biopsy
Inflammatory bowel disease (granulomatous, lymphocytic-plasmacytic, or eosinophilic enterocolitis), Mucosal lymphosarcoma, Amyloidosis	Histopathologic exam, absorption tests (supportive but nonspecific)
Dietary: abnormal fermentation	History
Neoplasms: lymphosarcoma, squamous cell carcinoma	Histopathologic exam
Peritonitis, abdominal abscessation	Peritoneal fluid analysis, ultrasound, exploratory surgery
Nongastrointestinal causes (chronic liver disease, congestive heart failure, renal disease)	Physical exam, clinicopathologic findings

Table 4 Differential diagnoses in chronic diarrhoea of adult horses in Equine Internal Medicine [7]

Differential Diagnoses for Acute Diarrhea in Adult Horses	
Common Causes	**Major Diagnostic Test(s)**
Salmonellosis	Fecal culture or polymerase chain reaction (PCR), culture of rectal mucosal biopsy
Clostridiosis *(Clostridium difficile, C. perfringens)*	Fecal culture, toxin analysis
Potomac horse fever *(Neorickettsia risticii)*	PCR (feces, peripheral blood), paired serologic tests
Antibiotic-associated diarrhea	History
Nonsteroidal anti-inflammatory toxicity (primarily right dorsal colitis)	History and supportive clinicopathologic findings, ultrasonography, exploratory surgery with biopsy
Undiagnosed	Other conditions ruled out
Less Common Cantharidin toxicity Parasitism (strongylosis, cyathostomiasis, other) *Aeromonas, Campylobacter* spp. Sand Carbohydrate overload Arsenic toxicity, other toxicities Thromboembolic disease Anaphylaxis	

Table 5 Differential diagnoses in acute diarrhoea of ault horses, in Equine Internal Medicine [7]

Summary of a Diagnostic Approach for Foals With Diarrhea

1. Physical examination*
2. Venous blood sample (complete blood count, chemistry panel)*
3. Fecal sample collection (flotation and infectious disease testing)*
4. Urine collection
5. Abdominal ultrasound
6. Abdominal radiographs

*These procedures and tests should receive priority

Table 6 Summary of the steps to reach a diagnostic in foals with diarrhoea, in **Equine Internal Medicine** *[7]*

Anex II

This annex is complementary information regarding the clinical case II. The lesions found in the intestine of the filly during the surgery were as follows.

All the visceras where in their correct anatomical positions, the small intestine had very poor motility and was covered in petechial hemorrhagic sites, with a very thickened wall. The caecum also presented a thickened wall was and very congested with most of its surface covered in petechial hemorrages. The large colon had a similar presentation, and in addition to that also presented at least five areas of full thickness necrosis, spread throughout the organ, each approximately 5x5cm, located in the folds of the sacculations of the ventral colon. The serosa was sloughing and peeling in these necrotic sites.

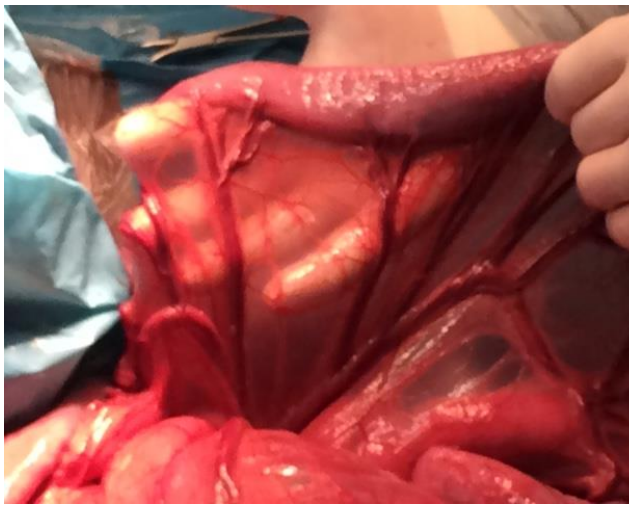


Figure 1 Small intestine looking congested and with thickened wall (with no motility)

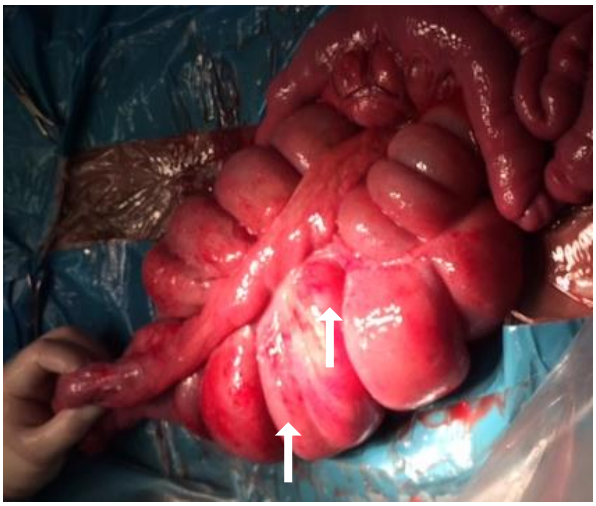
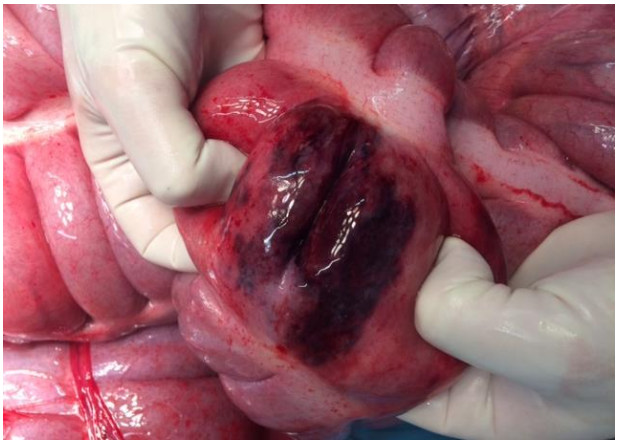


Figure 3 Large intestine Looking congested, with pethechia spread throughout its surface (white arrows)



Figures 4 and 5 showing necrotic areas spread throughout the large colon

The *post mortem* examination added other findings such as a very ulcerated mucosa in the small intestine, along with necrosis and complete replacement of mucosa with a diphtheric membrane in the distal half of the organ. The caecum had up to 3cm in depth of necrosis in areas in mucosal foals and so did the large colon, with large areas of the intestine showing severe transmural inflammation, with thrombosis, vasculitis and necrosis. Some kidney alterations were also found, such as hemorrhaging into the tubules and congestion of the organ. In the bacteriological exam ascarid eggs were found in moderate amounts, as well as heavy growth of *E.coli,* alfa-haemolitic streptococci and moderate growth of beta-haemolitic streptococci and yeast.
The lesions were consistent with severe necrotising enterocolitis.

This information was collected by observing the surgery as well as complemented by the *post mortem* examination report and the responsible veterinarian's report to the owner. All the pictures presented in this Anex were taken by myself, with the authorization of the hospital managers.